





trembling in 1; tremors in 3; cramps in 14; shivering in 2; great weakness in 15; headache and vertigo, 14—slight in 1, violent in 13; delirium in 4, slight in 2, violent in 2; occasional incoherence in 2; no delirium or sleepiness in 2; others not noted; pulse frequent, weak, and often imperceptible in 16, slow and irregular in 4, not mentioned in 32; lips blue in 2; countenance livid in 1; nails livid in 12; foaming at mouth in 2; sense of swelling of tongue or face, 13; of limbs, 1; hands clenched 2; syncope in 3; tenderness of epigastrium in 7; eyes glaring and protruded, 1, fixed in 1.

*Result.*—Recovered, 27; males, 9; females, 6; sex not given, 12. Died, 25; males, 14; females, 5; sex not stated, 5;—of these there died in one and a quarter hours, 1, a male; in two and a quarter hours, 1; in two and a half hours, 1, a female; in two hours, 8—males, 2, females, 2—sex not given, 4; in three hours, 4, males, 2, females, 1; no sex given, 1; in seven hours, 1, a male; in six days, 1, a male; in a short time, 2—1 male, 1 female; in a few hours, 1, a male; no time specified, 4, all males, five hours, 1, female.

*Post-mortem appearances.*—Lungs congested in 7; vessels of brain gorged in 5; mucous membrane of stomach red in 6; patches of darker color on its surface in 5; intestines injected in patches or otherwise in 6; rectum and œsophagus very red in 3; vessels of pia mater highly injected in 3; serous effusion under arachnoid in 4; at base of brain in 3; abdomen swollen in 2; bowels filled with air in 2; stomach empty in 2; containing gray-colored liquid in very small quantity in 3; filled with gas in 3; right side of heart filled with dark blood in 1; liver, spleen, and kidneys engorged in 1; healthy in 1; blood unusually fluid in 1.

(25.)

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ART. X.—*On the probable Cause of the Bruit, heard over the Heart in Acute Rheumatism.* By EZRA P. BENNETT, M. D., Ct.

I AM led from close and repeated observation to believe that in cases of acute rheumatism, the heart is not as often or as seriously implicated as many practitioners of the present day are inclined to believe. It is a fact not to be denied, that in a great majority of the cases of acute rheumatism, a bruit, more or less distinct, is plainly and distinctly to be heard. The question is, What produces it? It is generally conceded, I believe, that it depends upon some structural change in the valves, either thickening of the valves from inflammation or fibrinous deposits upon their free surfaces. That such causes of endocardial

murmurs do often exist is but too true. But that they always exist where these sounds are heard, or that they are the only cause of such sounds, I do most strenuously deny. In fact I do not believe that in the great majority of these cases there is any structural change in the valves whatever, or any deposits upon their surfaces. I believe the sounds in most of these cases depend entirely upon a change in the circulating fluid itself. We all know that in acute rheumatism, more than in any other disease, the plasticity and density of the blood is increased by the increased quantity of fibrin which it contains. Now this increased density and plasticity of the circulatory fluid must of course produce a decidedly increased friction upon the endocardial surface, and upon the valves, and in this way produce this endocardial murmur, in the same manner that an effusion of lymph, between the pericardium and the surface of the heart, produces the friction sound which we so plainly hear in pericarditis. Both these sounds, in my opinion, are produced in the same way, that is, by friction; one upon the internal surface of the heart, the other on the external. I have been led to adopt these views, in the first place, from noticing the little disturbance which takes place in the circulating system, during the existence of these complaints; and, secondly, from observing how quickly these sounds disappear, when the inflammatory state of the system is subdued, and the blood defibrinated, by proper remedies, which could not possibly be the case if these lesions actually existed. We know that a change of consistency in the circulating fluid does produce anæmic murmurs without any structural changes. Then why not in other diseases? The change in the blood in anæmia, though not identical with that in rheumatism, is, after all, analogous. In one fibrin is actually increased, in the other relatively so. In both cases the blood is more plastic, and the friction greater, and therefore both states of the blood are capable of producing endocardial friction sounds. My object, in this short communication, is to call attention to this view, which others may perhaps already entertain.



